Reprinted from Annals of The New York Academy of Sciences
Volume 111, Article 3, Pages 1049-1067
June 11, 1964

IMPLANTABLE CARDIAC PACEMAKERS*

Adrian Kantrowitz
Maimonides Hospital, Brooklyn, N. Y., and State University of New York Downstate Medical Center, Brooklyn, N. Y.

The commercial availability of implantable cardiac pacemakers in the past few years has considerably brightened the outlook of patients suffering from Stokes-Adams seizures with heart block. The mortality rate is steadily dropping with more extensive use of electronic devices for positive control of the heart rate over long periods of time.

The essential feature of Stokes-Adams seizures is a derangement of the rhythm of ventricular systole, varying from complete ventricular standstill to ventricular tachycardia and fibrillation. The resulting cerebral ischemia leads to a wide range of symptoms from slight dizziness and temporary unconsciousness to convulsions that are often fatal. The underlying heart block may be transient or permanent, partial or complete.

Since the incidence of heart block with Stokes-Adams seizures has been inadequately studied, only estimates are available. In reviewing 160,000 electrocardiograms taken at the Massachusetts General Hospital from 1925 to 1955 Rowe and White[1] discovered 350 patients showing complete atrioventricular dissociation, of whom 30 per cent had Stokes-Adams seizures. Penton, Miller, and Levine[2] reported 251 cases of complete heart block seen at the Peter Bent Brigham Hospital during a 42-year period, 61 per cent of them with associated Stokes-Adams seizures. At Maimonides Hospital (Brooklyn, N. Y.) 44 cases of Stokes-Adams syndrome were seen from 1956 through 1960 and 57 cases from 1961 to the present (August 1963), an increase probably reflecting our heightened interest in this problem. These figures suggest a country-wide incidence of heart block with Stokes-Adams seizures roughly paralleling that of congenital heart defects —estimated at 25,000 to 30,000 cases annually. If, as is generally agreed, arteriosclerosis is the main factor, the incidence of this combined disorder may rise in the near future. On the other hand, with better understanding of teratological defects in general, the incidence of congenital heart malformations may well decrease.

Treatment of intractable Stokes-Adams seizures by electrical stimulation of the heart was first suggested by the work of Callaghan and Bigelow[3] who carried out studies in animals with intact conducting tissues in 1951. A number of investigators have since then been interested in this method of controlling the heart rate. Zoll[4] in 1952 described an instrument for applying stimuli of rather high voltage via the skin, reporting successful results in patients with cardiac standstill, Weirich, Gott, and Lillehei[5] in 1957 used a myocardial electrode and an artificial pacemaker to control the heart rate in patients with surgically induced heart block.

Hopps and Bigelow[6] in 1954 showed that heart rhythm could be controlled by placing an electrode catheter in the superior vena cava and stimulating the region of the sinoatrial node. This concept was further developed in 1955 by Furman and Robinson[7] who passed an electrode catheter into the right ventricle and controlled the ventricular rate by means of an external electronic circuit. In 1959

* Basic work done at the Surgical Research Laboratory, Edward Neimeth Institute for Medical Research, Maimonides Hospital. Supported in part by Grant H-6510 from the U.S. Public Health Service.

Glenn, Mauro, and co-workers[8] reported successful treatment of Stokes-Adams disease with an implanted, externally powered electronic circuit. Chardack, Gage, and Greatbatch[9] in 1960 first reported on the use of an intrinsically powered circuit as a cardiac pacemaker. Our experimental efforts were started at about the same time, and a report of our early experience appeared in 1961.[10] Nathan and colleagues[11] in 1962 developed a totally implantable pacemaker that detects the normally generated P wave and fires the ventricle synchronously with the atrium. Schuder and Stoeckle[12] (1962) sutured a very small pacemaker receiver directly into the ventricle of dogs and paced the heart by inductive coupling of this unit with an external coil.

Long-term stimulation of the heart is thus effected by four principal means: (1) Implanted electrodes or an electrode catheter passed through the skin, with an external power source. The main disadvantages of this method are the high risk of infection from poor sealing of the wire or catheter as it passes through the skin, as well as the possible development of thrombosis, embolism, and/or thrombophlebitis from prolonged use of a catheter in a ventricular cavity and a vein. (2) An implanted receiver with an external power source. Chief among this system's drawbacks are the patient's need to have the cumbersome power source on his person day and night and to keep the transmitting unit precisely over the subcutaneously implanted receiver. Literal dependence of his heart on proper handling of the device tends to make the patient unduly preoccupied with his condition. (3) A self-powered, completely implantable, transistorized generator. The inherent disadvantage of these implanted devices is a limited battery life expectancy, now estimated to be 3 to 5 years. (4) A system of synchronous pacing, which also utilizes a self-powered unit. Its main drawbacks are rather extensive manipulation of the heart to place more than two myocardial leads and a highly complex electronic circuit. The purpose of employing an electronic pulse generator in treating patients with complete heart block and associated Stokes-Adams disease is to substitute a reliable electronic system for the heart's own impaired pacemaking system. Inasmuch as the main criterion is reliability, a relatively simple circuit is extremely important.

The Surgical Research Division of Maimonides Hospital and the Electronics Laboratory of the General Electric Company have jointly developed a totally implantable pacemaker with a fixed minimum rate, which also has an optional external control circuit for increasing the heart rate when desirable.

Design of the Pacemaker

The pacemaker consists of 5 batteries, 2 transistors, 3 resistors, and 1 capacitor —potted in an epoxy resin and hermetically sealed in a Silastic case. The unit weights 4 oz. and measures 1¼ × 4 × 6 cm.

The basic circuit consists of a pulse oscillator with a small number of components (FIGURE 1). It employs a PNPN complementary transistor configuration exhibiting negative resistance across the terminals of resistor R_1. The rationale of such a circuit was described by Suran[13] in 1956.

The two transistors act as a switch. When the capacitor (C) is discharged, the transistor switch is closed; C is thus charged from batteries E_1 and E_2. The current surge that passes through the heart is the stimulus. It takes about 2 msec. to charge C completely, after which the transistor switch opens and no longer conducts current. C takes about one second to discharge through R_1, thus determining the impulse frequency. Again the transistors act as a closed switch, and a second surge stimulates the heart for 2 msec. and so on. In this way we have a com-

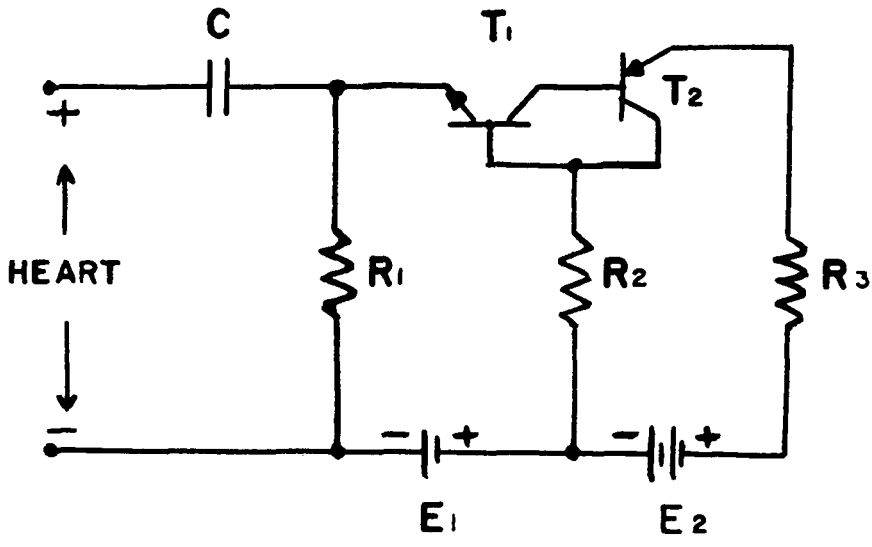

FIGURE 1. Basic electronic circuit of implantable pacemaker.

paratively simple, self-powered, reliable electronic circuit in a small package for implantation in the body.

Performance of the pacemaker circuit employing 5 1.4 volt mercury cells is as follows:

Peak pulse power—56 milliwatts
Peak output voltage—3.9 volts
Average current drain—25 microamperes
Average power drain—184 microwatts
Energy delivered to heart—64 microjoules
Power efficiency—38 per cent
Frequency—65 pulses/minute
Pulse time constant—2 milliseconds
Expected battery life—40,000 hours

Five models of the pacemaker have been produced by General Electric. Model I had a round Teflon case with essentially the same components as the present unit, and two Teflon-coated leads of braided, 49-strand, stainless steel wire.* It was used in our first three patients. We found it rather bulky and the exit point of the leads poorly designed. (In Case 1 a lead broke within three months and in Case 3, at 23 months.) Model II was rectangular and smaller; a redesigned exit point prevented sharp flexure of the leads. Twelve of these units were implanted. Model III had rounded corners, and the wire exit point was on the long side instead of the short side of the case. It was abandoned after 16 implantations because of several instances of wire breakage and two of fluid leakage into the case. Model IV's case and leads were given an additional coating of Silastic, and five units were implanted. In the present pacemaker, Model V's case is made of Silastic instead of Teflon. Since this material can be hermetically sealed, fluid

* 1.5 mil annealed type 316 stainless-steel wire. American Cyanamid Corp., Surgical Products Division, Danbury, Conn.

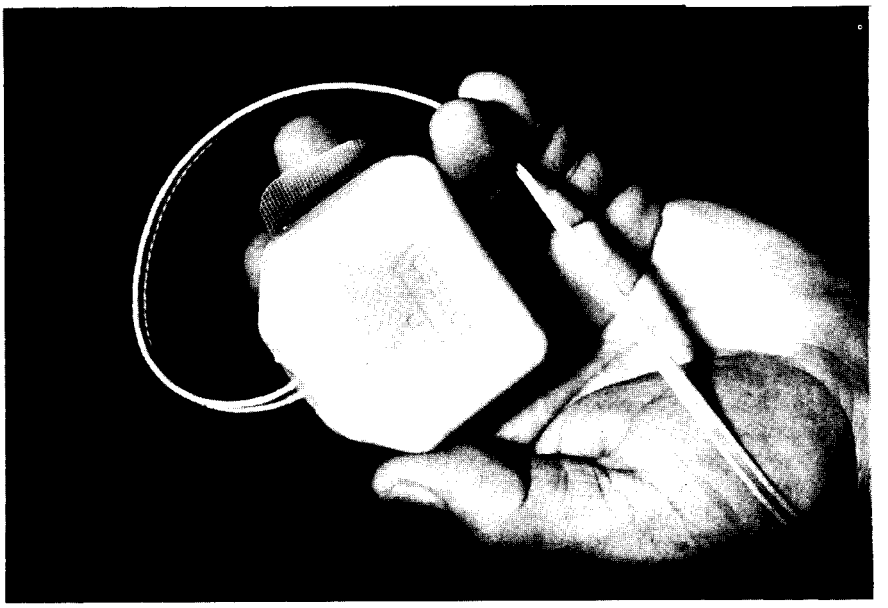

FIGURE 2. Model V pacemaker has Silastic case; twin lead is insulated with woven Dacron sleeve impregnated with Silastic.

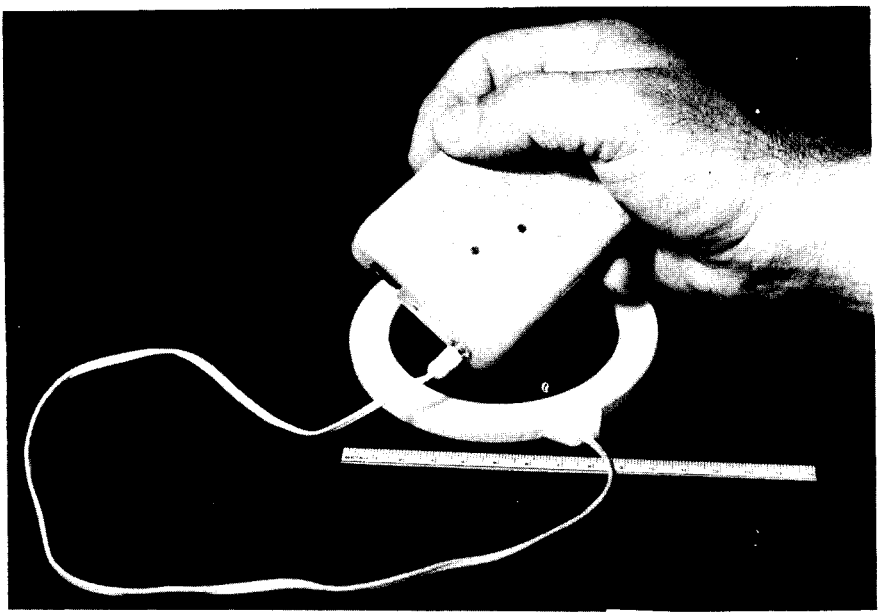

FIGURE 3. External control circuit.

leakage into the case should be eliminated. This pacemaker has a twin lead insulated with a woven Dacron sleeve impregnated with Silastic (FIGURE 2). The two electrodes are separated a few inches from the distal end for implantation in the myocardium. The twin lead's serviceability, revealed by GE's accelerated life tests, suggests excellent performance. A strip of Silastic-impregnated Dacron mesh bonded to the posterior surface of the case facilitates suturing of the unit to the anterior rectus sheath. Sixteen pacemakers of this type have been implanted. In all, 54 pacemakers have been implanted in 43 patients.*

The electronic circuit is so designed that it may be triggered by magnetic induction through the abdominal wall. The external control circuit (FIGURE 3) employs a PNPN complementary transistor configuration similar to that of the implantable circuit. The components are in an aluminum case measuring 9 × 6 × 1½ cm., which is connected by a 15 in. cable to a flat induction coil 3.4 in. in diameter. The coil is taped to the skin overlying the internal pacemaker. Because of its larger size, it is effective even if placed up to 1.5 in. off center.

The external control permits variation of the heart rate between the pacemaker's fixed rate (usually 64 pulses/min.) and 120 pulses/min. We believe this control circuit increases the pacemaker's usefulness with virtually no loss in reliability. A wire coil is merely wrapped several times around the components and soldered to the circuit at one point, before the case is sealed. Failure of this solder joint would not affect normal pacemaker functioning, but the rate could not be raised with the external control.

All the patients are paced at 80 to 90 pulses/min. via the external circuit during the first two postoperative days, and the rate is then gradually decreased over the next few days to the fixed minimum of 64. This is a safety measure for patients, many of them elderly, following thoracotomy when they would normally respond with a somewhat increased pulse rate.

The etiologic mechanism underlying Stokes-Adams seizures in 67 per cent of our patients has been ventricular tachycardia and fibrillation rather than cardiac standstill.[14] In several instances we have speeded up the pulse rate to suppress tachycardia and imminent ventricular fibrillation in the early postoperative period. Four patients were readmitted to the hospital, after recovery from the implantation procedure, with various intercurrent infections. The pulse rate in a patient who developed acute pyelonephritis, with temperatures up to 104° F, was kept at 80 or 85 via the external circuit until the infection was under control. In two patients with pneumonia and one suffering from a severe nose infection with a toxic reaction, a similarly increased heart rate also proved beneficial. Several of our younger patients, thoroughly familiar with the purpose and correct handling of the external control, increase their pulse rate as needed during periods of unusual physical or emotional stress.

Heart block with Stokes-Adams seizures was the indication for pacemaker implantation in 38 of our 43 patients. In the other five a slow heart rate had reduced cardiac output to the degree of frank congestive failure. Three of this group had heart block without Stokes-Adams seizures; one had the seizures without the block; and one had multiple arrhythmias with bradycardia. Their congestive failure was of the type responding best to a higher pulse. Four achieved a satisfactory rate with an implanted pacemaker. The fifth is much more comfortable with the pacemaker's basic rate raised to 85 by means of the external control.

* The second pacemaker in case 3 was implanted by Doctor Jack Cannon at UCLA Medical Center, Los Angeles, Calif.

The stimulus delivered by the pacemaker unit to the heart has been widely explored by ourselves and others.[5,9,15–18,21] The impedance of the load, i.e., the heart, as measured in three of our patients was approximately 300 ohms in series with a 20 microfarad capacitance.[22] Our early studies in animals and in humans showed that after the electrodes were in place and stabilized, approximately 15 microjoules of electrical energy was required to trigger the ventricle. We use a waveshape consisting of a sharp spike up to about 3.2 volts followed by a rapid subsidence so that about 90 per cent of the spike is over in 2 msec., even though it takes about 8 msec. for the voltage to return to zero (FIGURE 4). The unit's delivery of 64 microjoules gives a safety margin of over 300 per cent, which we have found adequate. Control of the amplitude of the impulse seems unnecessary with our pacemaker, since this amount does not appear harmful to tissue.

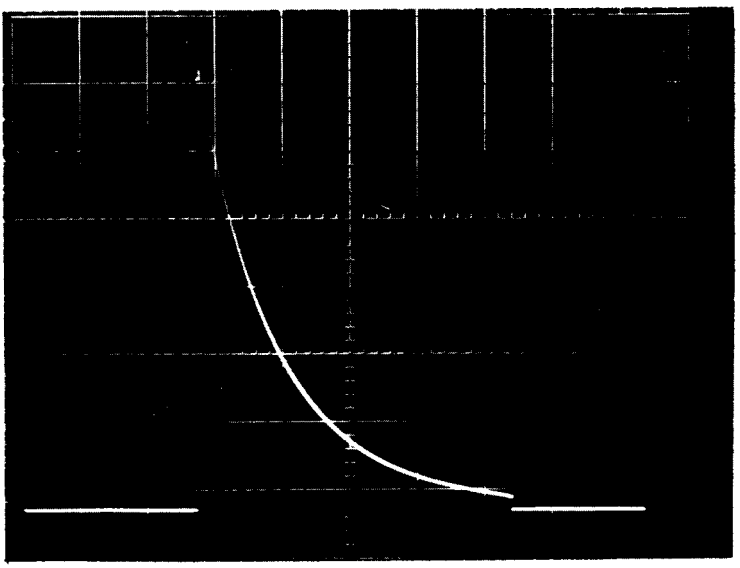

FIGURE 4. Output waveshape of implanted pacemaker. Abscissa calibration: 2 msec. per square. Ordinate calibration: 0.5 volt per square.

Surgical Technique

The operative procedure is carried out under general anesthesia with an endotracheal tube in place. All patients are taken to the operating room under the control of an external pacemaker with a cardiac electrode catheter passed through the right saphenous vein. Electrocardiographic leads are attached to the arms and legs, and the continuous ECG is displayed on an oscilloscope. The patient is placed in the dorsal recumbent position. Two incisions are made: a 6 cm. transverse incision beginning left of the umbilicus and deepened to the anterior rectus sheath and a left submammary incision. The pleural cavity is entered through the fourth intercostal space, and the pericardium is opened to expose the left ventricle. A subcutaneous tunnel connects the chest and abdominal incisions. The twin lead is passed up the tunnel and through the pericardium. Its two needle-equipped electrodes are separated and implanted in the myocardium with the

Silastic insulation fixed in the depths of the muscle. The bared wire is passed back through, emerging near the entry point of the insulated portion. At this point, a 3–0 silk suture fixes the bared wire to the insulated portion. A small metal flange on the bared wire prevents slipping. The needle is then removed. Details of the implantation technique are shown in FIGURE 5. The second electrode is implanted in the same fashion 1 cm. from the first. The pericardium is closed with interrupted silk sutures; the chest is closed in layers leaving an underwater drainage tube. The pacemaker is placed in the subcutaneous pocket and attached to the anterior rectus sheath by 3–0 silk sutures placed through the strip of Silastic-impregnated Dacron mesh bonded to the case. The subcutaneous tissues

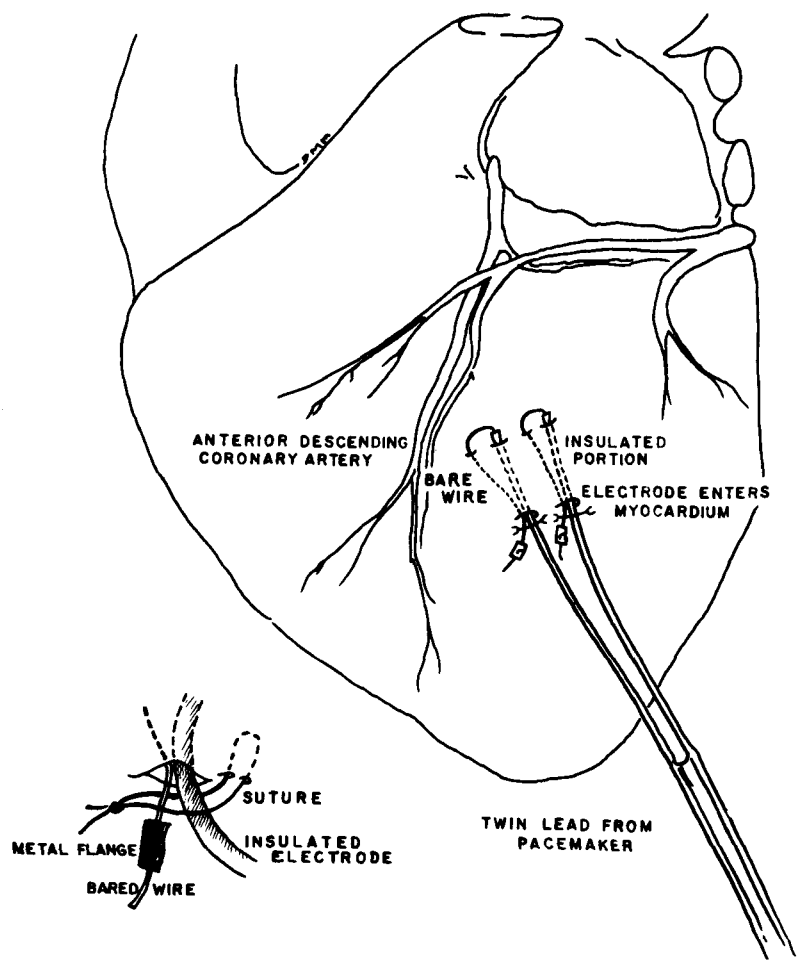

FIGURE 5. Details of suture technique. Electrodes are implanted 1 cm. apart in avascular area of left ventricle, with insulation fixed in myocardium. Bared wire is doubled back to entry point. Silk suture fixes bared wire, just below metal flange, to insulated portion of electrode (inset at left).

TABLE 1

IMPLANTATION OF PACEMAKER IN 43 PATIENTS

Case #, age, Sex	Indication for pacemaker	Implantation date, model	Postoperative complications	Outcome
1. R.C. 46–F				
2. S.K. 72–M	Heart block, Stokes-Adams seizures	5/5/61 Model I pacemaker		Died 5/9/61, diffuse coronary artery disease, congestive heart failure, and pulmonary edema.
3. N.A. 39–F				
4. M.S. 68–M				
5. M.R. 58–M				
6. B.A. 74–M				
7. P.B. 65–F	Heart block, Stokes-Adams seizures	9/13/61 Model II pacemaker		Alive and well.
8. J.H. 77–M	Heart block, Stokes-Adams seizures	9/26/61 Model II pacemaker	10/24/61 readmitted with signs of right heart failure. 150 ml. serosanguineous fluid removed from left pleural space. 9/6/62 treated medically for arteriosclerotic heart disease and paroxysmal dyspnea.	9/22/62 admitted as emergency to another hospital following acute myocardial infarction. Died one week later.
9. S.B. 61–F				
10. T.C. 74–F	Heart block, Stokes-Adams seizures	11/21/61 Model II pacemaker	At operation 200 ml. purulent fluid removed from pericardial sac.	Died 11/26/61, recent myocardial infarction demonstrated at autopsy.
11. R.L. 59–F	Sinus bradycardia, Stokes-Adams seizures	11/22/61 Model II pacemaker		Alive and well.

12. L.S. 82–M	Heart block, congestive heart failure (no Stokes-Adams seizures)	12/8/61 Model II pacemaker	3/27/62 readmitted for treatment of cellulitis of left nose with toxic reaction. External control kept at 80–85 until infection was under control.	Alive and well.
13. R.R. 69–F	Heart block, Stokes-Adams seizures	12/13/61 Model II pacemaker	2/6/62 congestive heart failure, treated by raising basic pacemaker rate with external control.	Alive and well.
14. J.S. 56–M	Heart block, Stokes-Adams seizures	12/28/61 Model II pacemaker		Died 1/3/62, recent myocardial infarction demonstrated at autopsy.
15. J.G. 55–F	Heart block, Stokes-Adams seizures	1/19/62 Model III pacemaker		Alive and well.
16. M.W. 60–F				
17. T.W. 69–M	Heart block, Stokes-Adams seizures	2/12/62 Model III pacemaker		Alive and well.
18. T.D. 85–F	Heart block, Stokes-Adams seizures	3/5/62 Model III pacemaker		Died 3/15/62, congestive heart failure.
19. E.G. 49–F				
20. M.V. 71–M	Heart block, Stokes-Adams seizures	6/11/62 Model III pacemaker	8/5/62 readmitted for treatment of acute cholecystitis. Cholecystectomy was performed. External control used to raise heart rate following surgery.	Alive and well.
21. Y.S. 65–F				
22. F.H. 78–F	Heart block, Stokes-Adams seizures	7/13/62 Model III pacemaker		Alive and well.
23. Z.B. 81–F	Heart block, Stokes-Adams seizures	7/19/62 Model III pacemaker		Alive and well.

TABLE 1 (continued)

Case #, age, Sex	Indication for pacemaker	Implantation date, model	Postoperative complications	Outcome
24. M.L. 70–F	Heart block, Stokes-Adams seizures	8/10/62 Model III pacemaker	1/12/63 readmitted with purulent discharge from submammary incision. Fistulous tract in chest wall excised.	Alive and well.
25. L.K. 70–M	Heart block, Stokes-Adams seizures	10/11/62 Model III pacemaker		Alive and well.
26. R.F. 68–M	Heart block, Stokes-Adams seizures	11/2/62 Model IV pacemaker		Alive and well.
27. F.H. 74–F	Heart block, Stokes-Adams seizures	11/5/62 Model IV pacemaker	11/22/62 readmitted, 700 ml. serosanguineous fluid removed from left pleural space.	Alive and well.
28. P.G. 76–M	Heart block, Stokes-Adams seizures	1/29/63 Model IV pacemaker	2/6/63 ventricular tachycardia and ventricular fibrillation.	Died 2/7/63, ventricular fibrillation.
29. E.P. 75–F	Heart block, Stokes-Adams seizures	3/5/63 Model V pacemaker		Alive and well.
30. S.B. 57–M	Heart block, Stokes-Adams seizures	3/7/63 Model V pacemaker		Alive and well.
31. M.C. 74–F	Heart block, Stokes-Adams seizures	3/21/63 Model V pacemaker		Alive and well.
32. P.S. 78–F	Heart block, Stokes-Adams seizures	3/29/63 Model V pacemaker	5/10/63 readmitted with cystitis. External control used to raise heart rate until infection was under control.	Alive and well.
33. L.M. 65–F	Sinus bradycardia, heart block (no Stokes-Adams seizures)	4/3/63 Model V pacemaker		Alive and well.

34. F.G. 63–F	Heart block, Stokes-Adams seizures	5/9/63 Model V pacemaker	After implantation, patient developed ventricular tachycardia which could not be controlled even though rate was raised to 102 by use of external control.	Died on operative day, ventricular fibrillation.
35. S.S. 63–M	Heart block, Stokes-Adams seizures	5/15/63 Model V pacemaker		Alive and well.
36. F.F. 78–F	Heart block, Stokes-Adams seizures	6/6/63 Model V pacemaker		Alive and well.
37. M.N. 66–M	Heart block, congestive heart failure (no Stokes-Adams seizures)	6/6/63 Model V pacemaker	Congestive failure relieved at basic rate of 64 but patient more comfortable at rate of 85 via external control.	Alive and well.
38. D.D. 72–M	Heart block, Stokes-Adams seizures	6/12/63 Model V pacemaker		Alive and well.
39. D.S. 63–F	Multiple arrhythmias, sinus bradycardia	6/19/63 Model V pacemaker		Alive and well.
40. B.E. 75–F	Heart block, Stokes-Adams seizures	6/24/63 Model V pacemaker		Died 6/26/63, congestive heart failure, mitral stenosis, coronary arteriosclerosis.
41. L.R. 81–M	Heart block, Stokes-Adams seizures	7/3/63 Model V pacemaker		Died 7/11/63, septicemia of unknown cause.
42. H.G. 81–M	Heart block, Stokes-Adams seizures	7/10/63 Model V pacemaker		Alive and well.
43. E.C. 68–F	Heart block, Stokes-Adams seizures	7/24/63 Model V pacemaker		Alive and well.

TABLE 2

ELECTROMECHANICAL FAILURES

Case #, age, sex	Indication for pacemaker	Implantation date, model	Postoperative complications	Treatment	Diagnosis confirmed	Outcome
1. R.C. 46–F	Heart block, Stokes-Adams seizures	9/28/60 two cardiac electrodes implanted and brought through skin; heart rate controlled by external pacemaker.				
		3/22/61 Model I pacemaker	6 weeks postoperatively readmitted with postcardiotomy syndrome.	Medical therapy		
			3 months after implantation pulse not following pacemaker; X-ray showed dislodged electrode.	Thoracotomy, electrode replaced in myocardium.	At operation	
			5 days later pulse again not following pacemaker. X-ray showed broken wire near exit from unit.	Abdominal wound opened. pacemaker replaced.	At operation	
		6/23/61 Model II pacemaker	21 months later ECG showed diminished amplitude of pacemaker spikes, pulse not following.	Abdominal wound opened, pacemaker replaced.	Failure due to battery exhaustion confirmed by GE.	
		3/14/63 Model V pacemaker				Alive and well
3. N.A. 39–F	Heart block, Stokes-Adams seizures	6/21/61 Model I pacemaker	22½ months later pacemaker rate over 100, pulse not following.	Abdominal wound opened, pacemaker replaced.*	At operation broken wire found near exit from unit.	
		5/3/63 Model V pacemaker				Alive and well

4. M.S. 68–M	Heart block, Stokes-Adams seizures	8/3/61 Model II pacemaker	3 weeks later postcardiotomy syndrome developed.	Medical therapy		
			6 months postimplantation, Stokes-Adams seizures recurred, slow pulse, no pacemaker spikes on ECG.	Abdominal wound opened, pacemaker replaced.	Case failure confirmed by GE.	
		2/21/62 Model III pacemaker	At 3-month check-up pacemaker rate was 500, regular sinus rhythm. Broken wire suspected.	None		Alive and well
5. M.R. 58–M	Heart block, Stokes-Adams seizures	8/9/61 two cardiac electrodes implanted and brought through skin; heart rate controlled by external pacemaker.				
		8/30/61 Model II pacemaker	2½ months later pacemaker rate over 100, pulse not following. X-ray showed broken wire.	Thoracotomy, pacemaker and wires replaced.	At operation	
		11/16/61 Model III pacemaker	2 months later slow pulse, pacemaker rate over 100, pulse not following.	Thoracotomy, pacemaker and wires replaced.	At operation wire break found, not shown on X-ray.	
		1/12/62 Model III pacemaker				Death 4 weeks postoperatively attributed to irreversible CNS damage from cardiac arrest during surgery.
6. B.A. 74–M	Heart block, Stokes-Adams seizures	8/30/61 Model II pacemaker	18 months later Stokes-Adams seizures recurred, pacemaker rate over 100, pulse not following.	Abdominal wound opened, pacemaker replaced.	At operation broken wire found, not shown on X-ray.	
		2/25/63 Model V pacemaker				Alive and well

* Surgery performed at UCLA Medical Center, Los Angeles, by Dr. Jack Cannon.

TABLE 2 (continued)

Case #, age, sex	Indication for pacemaker	Implantation date, model	Postoperative complications	Treatment	Diagnosis confirmed	Outcome
9. S.B. 61–F	Heart block, Stokes-Adams seizures	11/8/61 Model II pacemaker	9 months later Stokes-Adams seizures recurred, pacemaker rate over 100, pulse not following.	Thoracotomy, pacemaker and wires replaced.	At operation wire break found 3″ from myocardium.	
		8/14/62 Model III pacemaker				Alive and well
16. M.W. 60–F	Heart block, Stokes-Adams seizures	1/26/62 Model III pacemaker	1 year later pulse not following pacemaker, X-ray showed wire break.	Thoracotomy, pacemaker and wires replaced.	At operation	
		2/13/63 Model IV pacemaker				Alive and well
19. E.G. 49–F	Heart block, Stokes-Adams seizures	3/21/62 Model III pacemaker	5 months later pulse not following pacemaker. X-ray showed dislodged electrode.	Thoracotomy, electrode replaced in myocardium.	At operation	
			1 month later low amplitude pacemaker spikes on ECG, heart not following.	Thoracotomy, pacemaker and wires replaced.	Case failure confirmed by GE.	
		9/28/62 Model III pacemaker				Patient died 4 weeks postoperatively, acute ulcerative endocarditis with valvular perforation.
21. Y.S. 65–F	Heart block, Stokes-Adams seizures	6/15/62 Model III pacemaker	8 months later pulse not following pacemaker. Dislodged electrode suspected but not shown on X-ray.	Thoracotomy, electrode replaced in myocardium.	At operation	
			4 days later pulse again not following pacemaker.	Abdominal wound opened, pacemaker replaced.	At operation broken wire found	
		2/25/63 Model IV pacemaker				Alive and well

and skin are then closed. The external control unit is taped to the abdomen and set between 80 and 90 pulses/min.

Race *et al.*[18] and Klotz *et al.*[19] are among investigators who have made an intensive study of the optimum site for electrode implantation. We feel, however, that a comparatively avascular area on the anterior surface of the left ventricle has distinct advantages: it is readily accessible, and its wall is thicker and more muscular than that of the right ventricle, reducing the risk of entering the lumen during implantation of the electrodes. Each insulated lead is drawn into the myocardium for mechanical fixation and the bared wire is doubled back through the muscle for electrical contact. In this way the stress is well distributed.

Results

During the past 3 years the pacemaker has been implanted in 43 patients ranging in age from 39 to 85 years; 36 (or 84 per cent) had passed the 60 mark and 20 (or 46 per cent) were over 70. The unit is functioning well in 31 patients and, restoration of reliable, normal sinus rhythm in one other has made it unnecessary to replace his malfunctioning pacemaker. We feel that none of the 11 deaths in this series were related to failure of the pacemaker system. Early fatalities were attributed to recent myocardial infarction demonstrated at autopsy (2 cases), congestive heart failure (3 cases, in one of which pulmonary edema was also a factor), ventricular fibrillation (2 cases), and septicemia of unknown etiology (1 case). One patient died of central nervous system damage due to cardiac standstill that developed while a pacemaker was being replaced without insertion of an electrode catheter to assure adequate stimulation. The other two deaths were due to an acute myocardial infarction one year postoperatively and to acute ulcerative endocarditis associated with the patient's prolonged use of an electrode catheter inserted at another hospital. Our total experience is briefly reviewed in TABLE 1.

Electromechanical difficulties have occurred in nine patients. There were three such failures in our first case and two each in four other cases, bringing the total to 15. Wire breakage (9 instances) was by far the most common complication, an experience shared by other investigators who have reported their complication in detail. Levitsky and colleagues[20] encountered wire breakage in three of the eight patients studied. In Chardack's[9] series of 39 patients, wire breakage occurred in 10 (Hunter-Roth electrodes), local infections in 2, premature battery exhaustion and a faulty capacitor each in one case. Zoll[21] reported two instances of wire breakage and four local infections, one of them followed by electrode dislodgment, in a series of 21 cases.

Battery exhaustion occurred at 21 months in one of our patients. We have had no electronic failures. Fluid collected in two Model III pacemakers at about six months. We believe the tensile strength at the seal had been reduced by the high temperatures required for closure of the Teflon case. There were three instances of electrode dislodgment from the myocardium—at three, five, and eight months, respectively. At a regular check-up of one patient, his pacemaker was found to be nonfunctioning (a broken wire was suspected). Since he has normal sinus rhythm, no attempt has been made to replace the unit. Details of the electromechanical failures are shown in TABLE 2. We have had no complications of any kind with the Model V pacemaker during the six months it has been in use.

The General Electric Company has provided statistics on their total experience with the last three pacemaker models. About 500 units have been implanted, and accurate information is available on approximately half this number. Electronic

failure has occurred in 0.4 per cent, battery failure in 0.4 per cent, and case failure in 2.8 per cent (Model III only). Wire breakage has been a complication in 7.8 per cent of Model III and 0.8 per cent of Model IV pacemakers. This occurrence in 1.2 per cent of Model V units was probably related to poor placement of the leads. The cause of failure was undetermined in 1.2 per cent of Model III, 0.8 per cent of Model IV, and 1.2 per cent of Model V pacemakers.

After examining each faulty Teflon case and Teflon-coated lead returned to them, and consulting with leading workers in plastics, General Electric engineers concluded that failures of the case were due to the method of sealing. In the 15 leads studied the Teflon insulation had always fractured first, predisposing the wire to sharp flexure at this point.

Precise diagnosis of electromechanical failures has been a challenge to the engineers and medical people involved. Some failures can be easily pinpointed, and others resist the most exhaustive testing. In three of our patients a broken wire was clearly shown on X-rays, as the severed ends were separated. In such instances the pacemaker rate usually increases markedly, the ECG shows much weaker impulses, and the ventricular complexes do not follow the pacemakers. If the ends are barely separated, however, the fracture may not show up on X-rays. The ends may make contact when the patient moves, resulting in intermittent pacing of the heart.

A dislodged lead can usually be diagnosed by X-ray of the heart, the loop of the implanted electrode having opened. This was found in two of our three cases, and the loose lead was reimplanted. Three patients did not follow the pacemaker reliably because the heart's electrical energy requirement had increased appreciably. This complication in two of the three was related to a myocardial infarction in the electrode implantation area, with formation of fibrous scar tissue. It is well known that more electrical energy is needed to drive an impulse through this tissue than through the normal myocardium. The electrical resistance, per se, may be approximately the same as in the normal area, but the energy requirement is much higher. The onset of fibrosis may be detected during routine examination of a patient prior to the advent of actual failure. If the internal pacemaker is driven at a more rapid rate with the external control circuit, each pulse delivers a smaller amount of energy to the myocardium. The reduction is proportional to the rate: at 70 pulses/min., 64 microjoules is delivered and at 120 pulses/min., 30 microjoules. During the patient's postoperative visits we routinely check the pacemaker's ability to follow the external control up to 120 pulses/min. Failure to follow beyond 85 or 90 strongly suggests the development of fibrosis at the electrode implantation site or in the myocardium itself. The safety factor has thus been reduced considerably and the patient must be watched closely. Battery exhaustion, a faulty electronic component, or fluid leakage into the case causing a "short" is usually reflected by a diminution in amplitude of the impulse on the ECG or its complete disappearance. TABLE 3 summarizes the symptoms that General Electric engineers have found to be most frequently related to the failures occurring among the 250 pacemaker implantation on which they have reliable data. Some features of the chart may also be applicable to other types of pacemakers.

Summary

Complete heart block associated with Stokes-Adams disease now appears to be controllable by an electronic cardiac pacemaker. The advantages of totally implantable pacemakers are as follows: The operative procedure is not particularly complex; difficulties are rarely encountered if surgery is carried out with the

TABLE 3

AID FOR PACEMAKER DIFFICULTY

	1. Electronic (including failure of battery)	2. Battery discharge (or single cell failure)	3. Broken lead	4. Shorted electrodes	5. Subsequent heart damage (a later infarction)	6. Electrode pulled out of myocardium	7. Fibrosis: a. At electrode	7. Fibrosis: b. Interfering with lead wire	8. Case rupture	9. Foreign body reaction	10. Interference with other body functions	11. Case migration	12. Broken insulation on electrode lead wire	13. Pacemaker base rate too low
1. No stimulation occurring	□	△	□	□	△	△	○						△	
2. Heart returns to atrioventricular rate and or Stokes-Adams Syndrome	△	○	△	△	△	△	○						△	
3. Erratic stimulation		○	○		○								○	
4. No pacemaker output	□		△											
5. Small increase in pacemaker rate		△	○		△		△							
6. Large increase in pacemaker rate			△											
7. Pacemaker rate variable with, or responsive to: a. Patient motion			○	○		○							△	
7. b. Time (from one day to next)		△			△		○							
7. c. Patient position			○	○		○							△	
8. Heart rate higher than atrioventricular rate, but not following pacemaker		○	○		○	○	○						○	
9. Patient discomfort of: a. Soreness at pacemaker or lead wires								△	△	□				
9. b. Redness at pacemaker									△	△				
9. c. "Pulling" with patient motion								△			△	□		
9. d. Pacemaker visible or palpable dislocation								△				□		
9. e. Stimulation of other musculature (diaphragm or rectus muscles)			○			○					△		○	
9. f. Indications of infection									△	△				
10. Skipped stimuli		○	○		○	○		○					○	
11. Stimulus follows pacemaker at low rates, not at high rates		△			○	○		△					○	
12. Reduced pacemaker rate		○		□										
13. Patient in congestive failure														△

Key: probable □; likely △; and possible ○.

patient's heart under the positive control of an electrode catheter, introduced into the right ventricle via a superficial vein, and an external transistorized pacemaker. The postoperative course is usually smooth. A much clearer sensorium was noted in five of our older patients. The external control circuit has proved a valuable aid in a number of situations. Patients treated by an external pacemaker with wires or a catheter electrode passed through the skin are constantly concerned with having close at hand and protecting the unit that literally keeps them alive. On the other hand, patients with an implanted pacemaker, of which the main reminder is a moderate bulge on the abdomen, tend to forget about the problem. It is heartening to observe their changed outlook with improved cardiac output and release from the anxiety of impending Stokes-Adams attacks. (Typical elec-

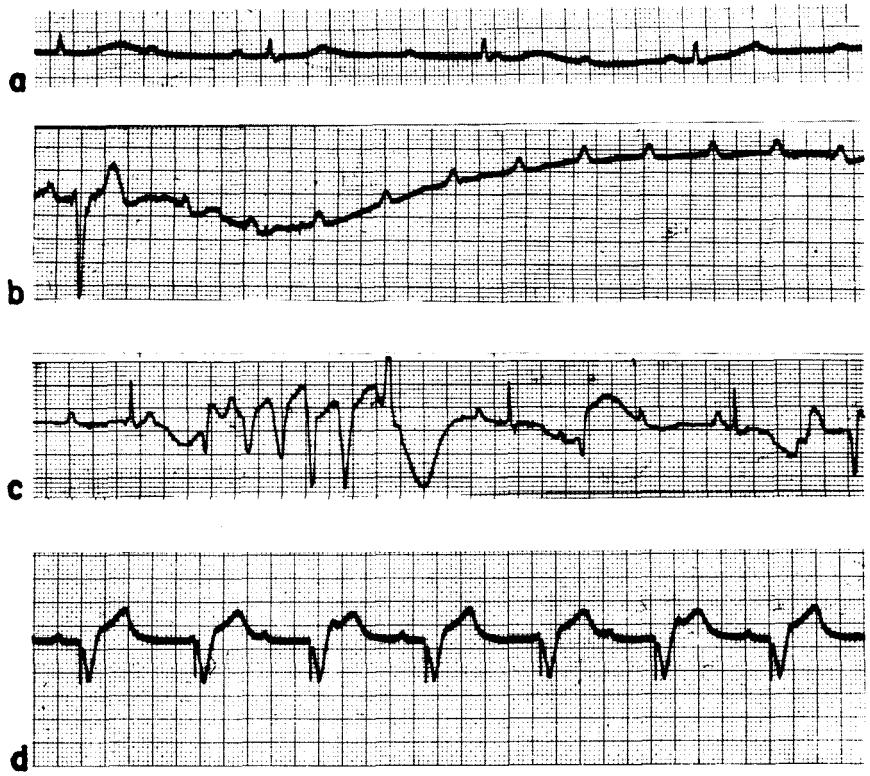

FIGURE 6. Typical ECG's of patients in this series: (*a*) complete heart block with idioventricular rhythm; (*b*) ventricular standstill; (*c*) complete heart block with ventricular tachycardia suggestive of imminent fibrillation; and (*d*) regular rhythm restored with an implanted electronic pacemaker.

trocardiograms of patients in our series appear in FIGURE 6.) With the new twin lead, which seems far more dependable than the earlier types, the pacemaker may well return to useful activity many persons who would otherwise succumb to heart block with Stokes-Adams disease.

Acknowledgment

The author is indebted to Mr. William D. Shadduck of the General Electric Company's X-ray Department, Milwaukee, Wis., for making available valuable technical data on the Company's total experience with the pacemaker.

References

1. ROWE, J. C. & P. D. WHITE. 1958. Complete heart block; a follow-up study. Ann. Intern. Med. **49**: 260.
2. PENTON, G. B., H. MILLER & S. A. LEVINE. 1956. Some clinical features of complete heart block. Circulation. **13**: 801.
3. CALLAGHAN, J. C. & W. G. BIGELOW. 1951. An electrical artificial pacemaker for standstill of the heart. Ann. Surg. **134**: 8.

4. ZOLL, P. M. 1952. Resuscitation of the heart in ventricular standstill by external electric stimulation. New Eng. J. Med. **247**: 768.
5. WEIRICH, W. L., V. L. GOTT & C. W. LILLEHEI. 1957. The treatment of complete heart block by the combined use of a myocardial electrode and an artificial pacemaker. Surg. Forum. **8**: 360.
6. HOPPS, J. A. & W. G. BIGELOW. 1954. Electrical treatment of cardiac arrest: A cardiac stimulator-defibrillator. Surgery. **36**: 833.
7. FURMAN, S. & G. ROBINSON. 1958. The use of an intracardiac pacemaker in the correction of total heart block. Surg. Forum. **9**: 245.
8. GLENN, W. W. L., A. MAURO, E. LONGO, P. H. LAVIETES & F. J. MACKAY. 1959. Remote stimulation of the heart by radiofrequency transmission. New Engl. J. Med. **261**: 948.
9. CHARDACK, W. M., A. A. GAGE & W. GREATBATCH. 1960. A transistorized self-contained, implantable pacemaker for the long-term correction of complete heart block. Surgery. **48**: 643.
10. KANTROWITZ, A., R. COHEN, H. RAILLARD & J. SCHMIDT. 1961. Experimental and clinical experience with a new implantable cardiac pacemaker. Circulation. **24**: 967.
11. NATHAN, D. A., S. CENTER, CHANG-YOU WU & W. KELLER. 1963. An implantable synchronous pacemaker for the long-term correction of complete heart block. Amer. J. Cardiol. **11**: 362.
12. SCHUDER, J. C. & H. STOECKLE. 1962. A micromodule pacemaker receiver for direct attachment to the ventricle. Trans. Amer. Soc. Artificial Internal Organs. **7**: 344.
13. SURAN, J. J. 1956. Circuit properties of the PNPN transistor. Proc. Nat. Conf. Aeronaut. Electronics.
14. DRESSLER, W., S. JONAS & A. KANTROWITZ. 1963. Observation in patients with implanted cardiac pacemaker. I. Clinical Experiences. Am. Heart J. **66**: 325.
15. ABRAMS, L. D., W. A. HUDSON & R. LIGHTWOOD. 1960. A surgical approach to the management of heart-block using an inductive coupled artificial pacemaker. Lancet. **1**: 1372.
16. CHARDACK, W. M. 1961. Correction of complete heart block by a self-contained and subcutaneously implanted pacemaker. J. Thorac. Cardiovasc. Surg. **42**: 814.
17. KANTROWITZ, A., R. COHEN, H. RAILLARD, J. SCHMIDT & D. S. FELDMAN. 1962. The treatment of complete heart block with an implanted controllable pacemaker. Surg. Gynecol. Obstet. **115**: 415.
18. RACE, D., G. R. STIRLING & P. EMERY. 1963. Electrical stimulation of the heart. Ann. Surg. **158**: 100.
19. KLOTZ, D. H., J. W. LISTER, S. L. JOMAIN, B. F. HOFFMAN & J. H. STUCKEY. 1963. Implantation sites of pacemaker following right ventriculotomy and complete heart block. J. Am. Med. Assoc. In press.
20. LEVITSKY, S., W. W. L. GLENN, A. MAURO, L. EISENBERG & W. P. SMITH. 1962. Long-term stimulation of the heart with radiofrequency transmission. Surgery. **52**: 64.
21. ZOLL, P. M., H. A. FRANK, L. R. N. ZARSKY, A. J. LINENTHAL & A. H. BELGARD. 1961. Long-term electric stimulation of the heart for Stokes-Adams disease. Ann. Surg. **154**: 330.
22. FELDMAN, D. S. & A. KANTROWITZ. 1963. Electrical characteristics of human ventricular myocardium stimulated in vivo. Clin. Res. **11**: 22.